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Genetic Etiology of Stability of Attention Problems in Young Adulthood

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Variation in attention problems in children and adolescents from non-clinical samples is highly heritable. It is unknown how attention problems develop later in life and whether the heritability in the general adult population is the same as in children and adolescents. We assessed the heritability and stability of individual differences in attention problems in the general young adult population and explored to what extent the stability can be attributed to genetic or environmental factors. On one or more occasions, young adult twins (age range, 18–30 years, $N = 4{,}245$) from the Netherlands Twin Registry filled out the attention problems (AP) subscale of the Young Adult Self-Report [Achenbach, 1997]: in 1991, N = 1,755 (of which 842 complete pairs), in 1995, N = 2,428 (1156 complete pairs) and in 1997, N = 2,344 (958 pairs). There was only a slight decrease in the average level of attention problems during young adulthood. The heritability at each occasion was around 40%. The correlation of attention problems across a period of 6 years was 0.42, and 77% of this correlation could be ascribed to genetic influences. Thus, individual differences in attention problems in young adulthood are heritable, and stability in individual differences over time can largely be ascribed to genetic influences. Genetic correlations across time were high, suggesting that the genes that influence variability in attention problems in late adolescence are largely the same as those that influence variability in early adulthood.

KEY WORDS: twins; adults; YASR; heritability; latent growth

INTRODUCTION

Principal component analyses on reports of common and less common behavior problems in the general population have shown that both in children and adults, a set of behavior problems can be identified that includes concentration problems and problems with finishing tasks. This cluster of problems has been termed attention problems [AP; Achenbach et al., 1995; Achenbach, 1997; Achenbach and Rescorla, 2001]. Studies on attention problems find that in childhood, more than half the variability in attention problems is heritable. Rietveld et al. [2004] for instance report a broad heritability (including non-additive genetic effects) of 75%, Schmitz and Mrazek [2001] report a heritability of 54%, and Edelbrock et al. [1995] report a heritability of 66% [see also Gjone et al., 1996; Schmitz et al., 1996; Zahn-Waxler et al., 1996; Hudziak et al., 2000, for similar estimates]. During childhood individual differences in attention problems as rated by the parents are relatively stable, in that scores correlate across measurement waves [Rietveld et al., 2004; Zukauskiene et al., 2004]. Correlation of attention problem scores between the ages of 7 and 12 can be mainly attributed to genetic influences [Rietveld et al., 2004, see also Larsson et al., 2004], whereas in adolescence, about half of the covariance is due to genetic influences [Van der Valk et al., 1998].

There are some studies that investigated the continuity of attention problems from adolescence to adulthood. Attention problems in adolescence are moderately correlated with attention problems in young adulthood and predictive of later psychopathology and maladjustment [Achenbach et al., 1995; Ferdinand and Verhulst, 1995; Ferdinand et al., 1995]. However, there has been no study that investigated the genetic background of this continuity into adulthood. And remarkably enough, no longitudinal study ever explicitly modeled the developmental course of attention problems in the general population: for example, do they generally diminish with age?

We report findings from a longitudinal data set on attention problems as reported by 4,245 twins between 18 and 30 years old from the general Dutch population in 1991, 1995, and 1997. The development of attention problems over time is studied in two ways. First, it is determined in what direction attention problems develop: do problems generally increase or decrease after adolescence, or are they largely stable? Second, we examine to what extent the stability of individual differences in attention problems over time can be ascribed to genetic or non-genetic sources, and whether attention problems in young adulthood are influenced by the same genes as during adolescence.

MATERIALS AND METHODS

Sample and Measures

Attention problems were assessed in 4,245 young adults from the Adult Netherlands Twin Registry [NTR; Boomsma et al., 2002]. Data were used from twins who on at least one occasion (in 1991, 1995, or 1997) filled out a postal survey. For a description of the population and response rates, see Boomsma et al. [2002]. Since we are interested in change in young adulthood and used a questionnaire designed specifically for 18 to 30-year-olds, data were used only from twins when they were between the ages of 18 and 30 (inclusive). This implies that if we have data from a twin pair in 1997, we ignore the 1997 data if by then the twins had reached the age of 31 and only use the information from the years in which they were younger than

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31. Similarly, if we have data on a twin pair in 1991, we do not include the data if the twins had not yet reached the age of 18 at that time, but only used the data from the years in which they were at least 18 years old.

Attention problems were assessed through the Young Adult Self-Report [YASR; Achenbach, 1997], of which the subscale attention problems (AP) contains seven items on: (1) concentration problems, (2) acting too young, (3) daydreaming, (4) being dependent on others, (5) having trouble finishing tasks, (6) being irresponsible, and (7) poor school/work performance. This subscale is normed for the age group 18–30 years. Subjects have to indicate the extent to which the item applies to them when taking into account the preceding six months: "not true," "somewhat or sometimes true," or "very true or often true". Counting "not true" = 0, "somewhat or sometimes true" = 1, and "very true or often true" = 2, the sum of the items is then used as a measure of the severity of attention problems. In 1991 and 1995, the surveys sent out to the twins contained an experimental version of the YASR. Because of this, one item from the AP subscale (on irresponsible behavior) was not present in 1991 and 1995. Below, we discuss how we dealt with this in composing a sum score.

Zygosity was determined by questionnaire items about physical similarity and frequency of confusion of the twins by family members and strangers or, when available, on DNA typing. For 30% of the same sex twin pairs, information on zygosity status is based on DNA polymorphisms, since they took part in other studies by the NTR. In this sample, agreement between zygosity status based on questionnaire data and zygosity based on DNA results was around 96%.

Data Analysis

The AP score as described above is a composite measure of seven ordinally scored items. In order to analyze the data, it would be convenient if we could treat the AP sum score as a metric trait and draw meaningful inferences about its variance. If we show that a sum score would provide the same information as a weighted composite score that takes into account the ordinal nature of the items, we can take a metric approach to the decomposition of the variance into genetic and non-genetic variance. A non-linear principal component analysis [PRINCALS; Van de Geer, 1993] was carried out on the data from a randomly drawn individual from each twin pair using the data from 1997. PRINCALS was used to determine the number of important dimensions underlying the seven items and whether factor scores based on a one-dimensional model would correlate with the sum scores. A perfect correlation (of +1 or −1) would indicate that when we use a linear approach in the analyses and model the sum scores directly, this leads to the same results as when we would include a non-linear measurement model in the structural modeling. It was also examined whether using only six items for a sum score would lead to a different phenotype. A perfect correlation between sum scores based on six and seven items would show that we were measuring the same phenotype and the results of the analyses based on only six items would be unbiased.

Subsequently, it was tested whether attrition in later years was in any way related to AP scores. Logistic regression analyses were performed, regressing attrition at the second and third wave on AP scores in the preceding wave(s). In order to correct for correlated observations, one twin was randomly chosen from each family. In addition, with these twins we tested whether the average score across waves was related to the number of datapoints in a one-way analysis of variance. Significant findings would indicate that the data are not missing at random. Additionally it was checked whether attrition was associated with zygosity status: monozygotic twins might be more motivated to continue with the research than dizygotic twins.

Next, several assumptions were tested regarding the equality of means, variances, and covariances across zygosity (five groups), sex, and measurement waves. This was done by fitting covariance matrices to the raw data using the Mx software [Neale et al., 2003] and comparing the maximized likelihood fit functions of nested models with increasing numbers of constraints. Nested models with particular restrictions on means and variances were compared with models without those restrictions in order to test the equality of parameters across zygosity, sex, or measurement waves. Significance of a test was determined by the difference in minus two times the log-likelihood (−2LL) functions of two hierarchically nested models, which is asymptotically χ^2 distributed with the degrees of freedom equal to the difference in degrees of freedom of the respective models. In order to reduce the probability of a Type II error (i.e., concluding that parameters are equal when they are not), a conservative overall α-level of 20% was maintained by testing each separate test at the Bonferroni corrected α/number of tests = 0.20/15 = 0.01 significance level.

A latent linear growth model [Meredith and Tisak, 1990] was fitted to study development of attention problems with age. Such a model involves the simultaneous estimation of linear regression lines for all subjects, regressing scores on measurement waves. The model postulates a normally distributed random variable slope for an individual's general change over time: an increase, a decrease, or no change in general degree of attention problems, defined as the number of score points increase per year. This is accomplished by fixing factor loadings from the 1991–1995–1997 scores on the latent slope variable to 0, 4, and 6 respectively, thereby taking into account the different time intervals between measurements. A covariate for sex was modeled onto the observed variables. There is an additional normally distributed random variable intercept that is used to define the arbitrary origin for each subject (here: in 1991) and is allowed to correlate with the slope variable. The model assumes that intercepts and slopes are normally distributed in the population. The estimated mean of the intercept is interpreted as the estimated mean of scores in 1991 and the estimated mean of the slope is interpreted as the estimated average direction of change over time. This way it can be assessed whether attention problems either increase or decrease with age, or are generally stable in young adulthood. The model was estimated using all available data, taking the different correlations between monozygotic and dizygotic twins into account.

Next, using quantitative genetic modeling, the heritability of individual differences was estimated and the source of stability over the years was determined. This modeling is based on the correlations between relatives that differ in the extent to which they are genetically similar and cross-correlations between these relatives for a trait measured at different times and consists of decomposing the variances and covariances, usually with structural equation modeling. Since monozygotic twins share all their genes and dizygotic twins on average only half their segregating genes, it is expected that when only additive genetic factors and non-shared environmental factors play a role, the correlation between monozygotic twins is twice the correlation between dizygotic twins. When non-additive genetic factors play a role, the difference between these correlations increases, and when shared environmental factors play a role, this difference decreases [Neale and Cardon, 1992].

The observed correlations between monozygotic and dizygotic twins did not suggest any environmental influences shared by twins from the same family or non-additive genetic effects, so a variance decomposition model was fitted that decomposed

TABLE I. Number of Participants at the Different Measurement Waves per Zygosity and Sex Status

	Individual twins					Twin pairs				
Zygosity/sex group	1991	1995	1997	Complete >1 wave	Complete at all waves	1991	1995	1997	Complete >1 wave	Complete at all waves
MZ male twins	266	385	379	275	66	128	185	164	121	29
DZ male twins	257	342	279	240	50	123	165	113	104	18
MZ female twins	401	602	639	464	115	192	289	273	203	43
DZ female twins	324	411	426	327	101	158	193	172	143	35
DZ OS										
Male twins	252	342	288	254	63	241	324	236	220	42
Female twins	255	346	333	278	67					

MZ, monozygotic; DZ, dizygotic; OS, opposite sex.

the (co-)variance matrix of the three measurements into an additive genetic part (A) and a non-shared environmental part (E). The A matrix then represents the (co-)variances that are shared 100% by monozygotic and shared 50% by dizygotic twins and E represents all (co-)variances unique to individual twins.

RESULTS

Descriptives

The numbers of twins and twin pairs with complete data on the six items (excluding irresponsibility) and between the ages of 18 and 30 years old on at least one measurement occasion are displayed in Table I. At the first wave in 1991, the ages of twins were between 19 and 25 with an average of 19.6 years (SD = 1.3). At the second wave in 1995, the ages were between 18 and 28 with an average of 21.3 years (SD = 2.4). At the third wave in 1997, the ages were between 18 and 30 with an average of 22.8 (SD = 3.1). The lowest observed sum score was 0, the highest 11 (highest possible: 12).

Non-Linear Principal Component Analysis

In order to study whether the seven items form an unidimensional scale, a PRINCALS analysis was carried out in a random subsample. The eigenvalues for a three-dimensional solution were 0.31, 0.15, and 0.14, respectively, indicating that there was only one major principal component. This result was replicated when using the remaining sample, with eigenvalues of 0.29, 0.15, and 0.14. Component loadings are presented in Table II. Apart from the relative sizes of the eigenvalues, the observation that the component loading patterns for the second and third components are not replicated provides further evidence of unidimensionality.

Next, using a unidimensional solution, individual factor scores were computed and compared with the sum scores based on seven items. A product-moment correlation of +0.99 was found (−0.98 in the replication sample), indicating that a sum score gives practically the same information as a composite measure that takes the ordinal nature of the items into account. Furthermore, sum scores based on seven items correlated 0.99 with sum scores based on six items (leaving out the item that was not in the 1991 and 1995 questionnaires), indicating that performing analyses on only six items would not significantly bias the results. Therefore, all remaining analyses were performed on sum scores based on the six items excluding the item on irresponsibility.

Testing for Selective Attrition

Randomly choosing one twin from each family, attrition at the second wave was not predicted by AP score at the first wave, $B = -0.02$, $\chi^2(1) = 0.21$. Similarly, attrition at the third wave was not predicted by AP score at the first wave, $B = 0.05$, $\chi^2(1) = 0.74$, nor AP score at the second wave, $B = -0.05$, $\chi^2(1) = 0.92$. Also, a one-way ANOVA showed no significant relation between the average score across waves and the number of data points, $F(2, 1186) = 0.49$. Attrition between 1991 and 1995 was not predicted by zygosity, $\chi^2(1) = 0.00$, nor was attrition between 1995 and 1997, $\chi^2(1) = 1.06$, $P = 0.30$.

Testing Assumptions About Equality of Means and (Co)-Variances

We estimated means and covariance matrices with dimensions 6 (3 waves × 2 twins) by 6 for each of the 5 zygosity/sex groups. The fit statistics for the hierarchically nested models are given in the Appendix. Means were equal across zygosity groups, but not across measurement waves and sexes (females scored higher than males, see Table III). Variances could be equated across zygosity groups, sexes, and waves. In addition, the autocorrelations were equal across zygosity groups and sexes: thus, the degree to which a 1991 score predicts a 1995 score is similar for both males and females and for monozygotic

TABLE II. Component Loadings for the Seven Items, once for the Randomly Drawn Sample of Twins, once for the Remaining Sample of Twins

	Random sample dimension			Replication sample dimension		
Item	1	2	3	1	2	3
Acting too young	0.37	0.57	0.60	−0.45	−0.14	−0.73
Concentration	0.69	−0.29	0.11	−0.65	−0.15	0.16
Daydreaming	0.55	−0.48	0.11	−0.50	−0.49	0.16
Poor school/job performance	0.63	0.02	−0.42	−0.54	0.34	0.50
Irresponsible	0.43	0.61	−0.33	−0.49	0.51	−0.38
Dependent	0.57	−0.09	0.43	−0.49	−0.50	0.02
Fails to finish	0.63	0.04	−0.32	−0.64	0.34	0.09

TABLE III. Estimated Variance, Means and Correlations Based on the Final Model (See Text)

	Estimate	95% confidence interval
Variance of scores	3.98	3.83–4.14
Mean scores for females		
Wave 1	3.64	3.52–3.77
Wave 2	2.69	2.59–2.80
Wave 3	3.06	2.95–3.16
Mean scores for males		
Wave 1	3.17	3.02–3.31
Wave 2	2.53	2.41–2.65
Wave 3	2.72	2.59–2.84
Autocorrelations		
Wave 1, wave2	0.46	0.41–0.50
Wave 2, wave 3	0.58	0.54–0.61
Wave 1, wave 3	0.42	0.36–0.48
MZ correlation		
Equal across waves	0.40	0.36–0.45
DZ correlation		
Equal across waves	0.19	0.14–0.24
MZ cross correlations		
Wave 1, wave 2	0.31	0.24–0.39
Wave 2, wave 3	0.38	0.31–0.44
Wave 1, wave 3	0.34	0.25–0.42
DZ cross correlations		
Wave 1, wave 2	0.09	0.02–0.16
Wave 2, wave 3	0.16	0.10–0.22
Wave 1, wave 3	0.12	0.03–0.20

MZ, monozygotic; DZ, dizygotic.

and dizygotic twins. Opposite sex twin correlations equaled same sex correlations in both males and females from dizygotic twin pairs: that is, a male and a female from an opposite sex twin pair are just as similar as two males from a dizygotic twin pair or two females from a dizygotic twin pair, suggesting that the same genetic factors are operating in males and females. Both monozygotic and dizygotic twin correlations were equal across waves, which means that similarity between individuals from a twin pair is not dependent on the year of testing. Cross-twin, cross-wave correlations were also equal across zygosity/sex groups. The within-wave twin correlation for monozygotic twins was significantly larger than the correlation for dizygotic twins, suggesting that the trait is heritable. Table III gives the parameters for the final model. The dizygotic twin correlation and cross correlations are about half the monozygotic correlation and cross-correlations, indicating that only additive genetic effects and non-shared environmental factors explain individual differences in attention problems.

The autocorrelations between the three waves are significantly different, $\chi^2(2) = 31.33$, $P < 0.001$, suggesting developmental changes in the ranking of individual AP scores. The autocorrelations indicate a longitudinal structure where the correlation between measurements is inversely related to the distance in time.

General Direction of Developmental Change

The fitting of a latent linear growth model to describe individual developmental change over time yielded a −2LL of 26631.36 with 6,501 degrees of freedom (26 estimated parameters). The mean of the slope, which indicates the overall average change over time, was significantly different from zero and estimated at −0.09 points per annum (95% CI: −0.11, −0.07), indicating a general decrease of attention problems with age.

Decomposition of (Co)-Variances

In order to explore the operation of genetic and environmental factors in stability, the covariance structure was decomposed into genetic and environmental covariance structures. The matrices for additive genetic effects A and non-shared environmental effects E are presented in Table IV. The additive genetic component could not be dropped. Its omission led to a significant decrease in model fit, $\chi^2(6) = 55.45$, $P < 0.0001$. The estimates on the diagonals indicate that at each wave, the relative influence of genetic variability is smaller than the relative influence of (non-shared) environmental variability. The heritability is defined as the proportion of the total variance explained by additive genetic variation. Thus, we estimate the heritability at the first wave as $1.68/(1.68 + 2.52) = 0.40$. The heritability estimates for wave 2 and 3 are 0.37 and 0.44, respectively. In a similar fashion, we can compute the proportion of the (co-)variance shared by wave 1 and wave 3 as $1.31/(1.31 + 0.40) = 0.77$: 77% of the stability of individual differences over a period of 6 years can be attributed to genetic individual differences. Thus, the observation that one individual generally scores lower than another individual is better explained by genetic factors than by environmental factors. Evidently, a large proportion of the influence of non-shared environmental factors is time-specific and/or consists of measurement error.

TABLE IV. Estimated Covariance Matrices for Additive Genetic Variance (A) and Non-Shared Environmental Variance (E)

Wave	A			E		
1	1.68			2.52		
2	1.14	1.47		0.76	2.50	
3	1.31	1.44	1.67	0.40	0.79	2.15

By standardizing A and E, the genetic and environmental correlations are obtained (see Table V). The genetic correlations are high, indicating that at the different measurement waves, largely the same genes contribute to individual differences in severity of attention problems. In contrast, the environmental correlations are low, indicating that different environmental factors operate at different points in time. In sum, these data suggest that the same genes but different environmental factors are operating across time, and most of the stability in attention problems can be ascribed to genetic effects. Note that this is not necessarily the case: it is possible that the exact same genes operate across time, but contribute only little to stability over time (that is, when heritability is low).

DISCUSSION

The genetic background of attention problems was studied in a large group of young adult twins between 18 and 30 years old over a period of 6 years. We did not have complete data for every twin, but analyses showed that these data could be assumed to be missing at random. Heritability estimates for attention problems were around 40% at each measurement wave, and are lower than the estimates reported earlier for children. There was no evidence of non-additive genetic effects. There are several possibilities for these differences in heritability across age groups. First of all, we might be dealing with a slightly different phenotype: the AP subscale items of the YASR are not the same as the AP subscale items on the Child Behavior Checklist [CBCL; Achenbach and Rescorla, 2001]. In addition to some of the adult items, the child AP subscale includes the items "can't sit still," "impulsive," "confused," "inattentive," and "stares." The young adult items "dependent" and "irresponsible" are not present in the child version. Moreover, in child studies the CBCL is usually filled out by parents and teachers, whereas the YASR concerns self-reports. Attention problem scores based on self-reports are usually lower

TABLE V. Genetic and Environmental Correlations Over Time (95% Confidence Intervals in Parentheses)

Wave	Additive genetic			Non-shared environmental		
1	1			1		
2	0.73 (0.57–0.89)	1		0.30 (0.20–0.40)	1	
3	0.78 (0.59–0.97)	0.92 (0.81–1.00)	1	0.17 (0.04–0.40)	0.34 (0.26–0.42)	1

Correlation matrices are obtained through standardization of matrices **A** and **E** from Table IV.

than scores based on parent-reports [Zukauskiene et al., 2004].

These differences regarding both items and mode of reporting across measurement instruments may also have contributed to the higher scores for females than for males, which has been reported before in young adults [Achenbach and Rescorla, 2003] and disagrees with the findings in children, where boys tend to score higher than girls [Rietveld et al., 2004].

On average, attention problems decrease in severity during young adulthood but at a rate of only 0.09 of a score point per annum. Thus, no dramatic changes are observed after adolescence and AP scores remain largely at the same level. AP scores are correlated across waves. The question is whether this stability of individual differences can be attributed to genetic factors or environmental factors, or both. The time-specific heritabilities are around 40%. When looking at the variance that is stable over time (i.e., the variance that the multiple measurements had in common), the heritability increases. A large part of the variance in one measurement can be attributed to environmental influences that are time-specific (including measurement error) and that do not contribute to stability in individual differences over time. Genetic factors explain the correlations across time much better. We found that stability of individual differences over a period of 6 years could for 77% be attributed to genetic factors. High genetic correlations (0.73–0.92) indicate that largely the same genes explain individual differences across time. The fact that most respondents were about 18 years old at the beginning of this longitudinal study therefore allows us to conclude that the genes that are responsible for variability in attention problems in young adulthood are the same as those responsible for variability in late adolescence.

The Department of Biological Psychology at the Vrije Universiteit in Amsterdam has been collecting data on problem behavior longitudinally in children since 1986 and will continue to do so. Future studies will look into the stability of attention problems in the general population from early childhood (parent and teacher reports), through adolescence (parent, teacher, and self-reports) into adulthood (self-reports) and investigate differential developmental trajectories where some children show persistence of problems well into adulthood, whereas others show remission. Individual differences in developmental trajectory might well have a genetic component. Moreover, developmental trajectories with attention problems persisting into adulthood will probably be associated with comorbid conditions and future analyses will therefore take into account their moderating effects.

An important issue to be addressed in these future analyses is measurement invariance. In the general population, behavior problems related to inattention and hyperactivity in adulthood seem to cluster in a different way than in childhood [Achenbach, 1997; Achenbach and Rescorla, 2001] and attention-related problems are therefore measured with only partly overlapping sets of items. Furthermore, self-reports are not available from young children and parent reports are usually not available in adulthood. Development from childhood into adulthood is therefore more difficult to study and needs to be addressed using more sophisticated methods that model development at the item level.

APPENDIX

Test Results for the Equality of Means, Variances, and Covariances

Model	Test	# Parameters	−2LL	Df	Comparison model	χ^2	df	*P*
1	(Saturated model)	87	26440.42	6,440				
2	Means equal across zygosity groups	75	26453.24	6,452	1	12.81	12	0.38
3	Means equal across sexes	72	26489.09	6,455	2	35.85	3	<0.01
4	Means equal across waves	71	26644.82	6,456	2	191.58	4	<0.01
5	Variances equal across zygosity	63	26464.58	6,464	2	11.34	12	0.50
6	Variances equal across sexes	60	26465.88	6,467	5	1.30	3	0.73
7	Variance equal across time	58	26470.70	6,469	6	4.81	2	0.09
8	Autocorrelations equal across zygosity	46	26485.03	6,481	7	14.33	12	0.28
9	Autocorrelations equal across sexes	43	26485.57	6,484	8	0.54	3	0.91
10	SS twin correlations equal across sexes	37	26488.23	6,490	9	2.66	6	0.85
11	SS DZ twin correlations equal to OS DZ twin correlations	34	26494.01	6,493	10	5.77	3	0.12
12	SS cross correlations equal across sexes	28	26499.63	6,499	11	5.62	6	0.47
13	DZ OS cross-correlations are symmetrical	25	26502.21	6,502	12	2.58	3	0.42
14	DZ OS cross-correlations equal to DZ SS cross correlations	22	26504.56	6,505	13	2.35	3	0.50
15	twin correlations equal across time	18	26508.88	6,509	14	4.32	4	0.36
16	MZ correlation equals DZ correlation	17	26548.80	6,510	15	39.93	1	<0.01

DZ, dizygotic; MZ, monozygotic; SS, same sex; OS, opposite sex; cross correlation, cross-twin cross-wave correlation.

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